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JOHN SPELLMAN
Governor

ALAN J. G
Secretary

STATE OF WASHINGTON
DEPARTMENT OF SOCIAL AND HEALTH SERVICES
Olympia, Washington 98504

August 25, 1983

Joel Mulder
Toxic Waste Management
215 Fremont
San Francisco, CA 94105

Dear Joel:

The recently announced NESHAP for arsenic is of interest to many Washington State residents because of its implications for the ASARCO smelter in Ruston. At an August 16 public meeting at Tacoma's Wilson High School, the EPA explained the epidemiological evidence supporting the public health risk analysis for community ambient arsenic exposure. This risk analysis was based on three factors: 1) exposure estimates for arsenic (based on a computer model of emissions), 2) unit risk estimates (based on epidemiological studies of smelter worker populations), and 3) population counts from the Bureau of the Census. A major concern here relates to the unit risk estimates which predict the health risk associated with lifetime exposure to 1 microgram per cubic meter of ambient arsenic. If my analysis is correct, these estimates may be flawed. Since public policy for both the NESHAP and much of the proposed Superfund cleanup will be based on this risk assessment, I am requesting a CDC review of the methodology on which the assessment is based.

During the Wilson High School meeting, a Ruston resident questioned the evidence supporting a linear dose-response relationship between arsenic exposure and lung cancer risk. He noted that although the observed number of deaths increased with increasing exposure, so did the expected number of deaths. This same problem has plagued epidemiologists working on arsenic, since the relative risk remains about 2 for the different exposure groups. (The relative risk equals the ratio of the observed to the expected number of deaths.)

Instead of using the relative risk to measure association of exposure and disease, the EPA has opted to use an Absolute-Risk model. This model calculates the difference between the observed and expected number of deaths and divides by the number of person-years of observation.

Relative Risk	Absolute Risk
Observed/Expected	(Observed-Expected)/(Person-years)

During the meeting, I pondered why the Absolute-Risk model would show a dose-response relationship while the more traditional measure, the Relative Risk, showed no relationship. Applying a little simple algebra to the models gives the following. Everyone seems to agree that for each exposure group (highest to lowest) the relative risk is approximately equal to "2". Thus Observed Deaths = 2 x Expected Deaths. Substituting this value into the Absolute-Risk model gives the following result:

$$\frac{\text{Observed} - \text{Expected}}{\text{(Person-years)}} = \frac{(2 \times \text{Expected}) - \text{Expected}}{\text{(Person-years)}} = \frac{\text{Expected}}{\text{(Person-years)}}$$

In using this formula, it must be kept in mind that age is a confounding factor. Older cohorts have a higher ratio of expected cancer to person-years of exposure because older people are expected to (and do) get cancer more frequently than do younger people. According to Dr. Enterline and Dr. Milham, the groups with the highest dose exposure are people exposed many years ago (and therefore older). Better controls at smelters have more recently reduced the level of arsenic exposure to workers. Thus the workers with the highest level of exposure are expected to have an increased incidence of cancer merely because of their age. This fact could explain the linear relationship between the absolute risk [Expected/(Person-years)] and exposure dose. The same relationship of age and exposure would not confound the relative-risk measure.

If my analysis is correct, the relationship of dose and cancer risk which supports the NESHAP may be nothing more than a relationship between age and risk of cancer. The actual data support a causal relationship of worker exposure to arsenic but do not support a dose-response relationship. The implications of this analysis for regulation could be substantial. However I assume that my analysis is flawed, since such a mistake by the EPA seems unlikely. I've discussed the problem with the EPA Risk Assessment group but I am no closer to identifying my mistake. I appreciate your efforts in reviewing the analysis and hope the review will lead to a better understanding of the risks of community ambient arsenic exposure.

Sincerely,

Floyd Frost

Floyd Frost, Ph.D.
Chronic Disease Epidemiologist
Division of Health, B17-9

FF:cb

cc: John Beare, M.D.
Samuel Milham, Jr., M.D.
Jack Allard
John Spencer
Earnesta Barnes

JOHN SPELLMAN
Governor

ALAN J. GIBBS
Secretary

STATE OF WASHINGTON
DEPARTMENT OF SOCIAL AND HEALTH SERVICES
1409 Smith Tower, B17-9 • Seattle, Washington 98104

August 31, 1983

Joel Mulder
Toxic Waste Management
215 Fremont
San Francisco, CA 94105

Dear Joel:

The recently announced NESHAPS for arsenic have sparked a lively debate over the ability of health effects studies to detect arsenic-induced lung cancer among Tacoma residents. Because the sensitivity of such studies has not been examined relative to the model of excess risk, postulated by the EPA, the continuing debate is based on personal opinions. As you might imagine, the opinions of the various officials differ on this matter.

An alternative to this debate is to statistically examine the sensitivity of the existing studies. I am requesting funding for such an analysis which will further our understanding of the limits of detection for an excess lung cancer risk among Tacoma area residents.

In a recent public meeting in Tacoma, the EPA announced that four additional lung cancer deaths are expected each year in the Tacoma area from ASARCO arsenic emissions. With a background level of 270 lung cancer deaths each year in the impact area, this increase would almost certainly go undetected. However, a closer inspection of the EPA model suggests effects on sub-populations which may be detectable. For example, the model postulates no interactive effect of arsenic exposure and smoking. Thus, almost three of the four excess lung cancer deaths would be among non-smokers (assuming 33% of the population smokes). Since lung cancer among non-smokers is extremely rare (only 17 deaths per 100,000 people, age 35-84, per year), an increase of three deaths per year may be detectable.

Although we do not know which lung cancer deaths were related to smoking, we know that women have not, until recently, consumed their fair share of cigarettes. Consequently, they have not suffered much lung cancer. Between 1950 and 1970, only 146 Tacoma women died of lung cancer (6.8 deaths per year). Since half of the postulated excess lung cancer deaths, according to the EPA model, would occur among women, and since women are also less likely to have confounding occupational exposures, this group would provide a sensitive test to detect arsenic-induced lung cancer.

For the years 1950 to 1970, a DSHS study examined lung cancer by census tract for Tacoma. I propose to examine the ability of this historical lung cancer mortality study to detect an excess number of community lung cancer cases. We will statistically test whether .5, 1, 1.5, etc., additional female lung cancer cases can be detected given the expected number of cases for the time period and population at risk. The analysis would require the following data:

1) the coding of 199 female lung cancer deaths (which occurred between 1970 and 1975) in Pierce County to census tract. Lung cancer deaths are already coded by DSHS to census tract for the period 1950 to 1970.

2) the pattern of arsenic exposure by census tract (obtained from the EPA). This will allow us to calculate what fraction percent of the postulated excess deaths occur in each census tract.

3) urban-female age-specific lung cancer mortality rates for 1950, 1960, 1970 and 1980 for the U.S.

4) census data for females for 1950, 1960, 1970 and 1980 by age (ages 35-84) and by census tract for Pierce County.

I believe the above analysis would go a long way toward resolving the issue of the ability of health studies to detect an excess of lung cancer in Tacoma. Attached is a budget covering the costs of conducting the analysis outlined above. I appreciate your efforts in exploring funding sources for such a study.

Sincerely,

Floyd Frost

Floyd Frost, Ph.D.
Chronic Disease Epidemiologist

FF:bl

Enclosure

cc: John Beare, M.D.
Samuel Milham, Jr., M.D.
J. Allard, Ph.D.
John Spencer
Ernesta Barnes

BUDGET

Salaries	
Research Analyst 3 2 months at $2,000	$4,000.00
Indirect Costs	712.00
Fringe Benefits	840.00
Facilities (rent)	260.00
Cost pool (supplies)	310.00
Travel	100.00
Computer time	300.00
Purchase of census data	460.00
Coding of census tracts	125.00
TOTAL BUDGET	$7107.00